WEST Search History

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DATE: Thursday, December 09, 2004

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		DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L7	(oestro$ or oestra$ or estro$ or estra$) adj10 (plasma adj1 cholesterol)	18
☐	L6	(oestro$ or oestra$ or estro$ or estra$) adj5 (plasma adj1 cholesterol)	9
☐	L5	(oestro$ or oestra$ or estro$ or estra$) adj5 cholesterol	371
☐	L4	(oestro$ or oestra$ or estro$ or estra$) same cholesterol	1440
☐	L3	(oestro$ or oestra$) same amyloid$	12
☐	L2	(oestro$ or oestra$)	6417
☐	L1	(oestro$ or oestra$) adj10 amyloid$	0

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L6: Entry 5 of 9 File: USPT Nov 27, 2001

DOCUMENT-IDENTIFIER: US 6323190 B1
TITLE: Estrogen mimetics lacking reproductive tract effects

Other Reference Publication (21):
Lundeen et al., "Characterization of the Ovariectomized Rat Model for the Evaluation of Estrogen Effects on Plasma Cholesterol Levels," Endocrinology, 138 (4):1552-1558 (1997).

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L6: Entry 8 of 9 File: USPT Nov 30, 1999

DOCUMENT-IDENTIFIER: US 5994337 A
TITLE: Effects of 17.alpha.-dihydroequilenin on plasma lipid and lipoprotein, glucose, insulin concentrations, coronary artery vasomotor function, and reproductive organ and mammary gland proliferation in atherosclerotic mammals

Brief Summary Text (2):
Postmenopausal estrogen replacement therapy has gained wide recognition as a lifelong preventive regimen for the reduction of osteoporotic fracture and ischemic heart disease. Unfortunately, good scientific evidence has failed to persuade the majority of menopausal women that the benefits of long-term estrogen replacement therapy are worth the inconvenience or anxiety resulting from its side effects, especially vaginal bleeding and the putative increase in breast cancer risk. Additionally, most of the evidence of estrogen effects in preventing ischemic heart disease in the United States is from studies that used unopposed conjugated equine estrogens. The current evidence is unclear as to whether the addition of progestins, necessary to prevent iatrogenically induced endometrial carcinoma, may either partially or completely negate the cardioprotective effect of unopposed estrogens. Several of the inventors have described a component of Premarin.RTM. (Wyeth-Ayerst, Princeton, N.J.), 17.alpha.(-dihydroequilenin (DHEN), that caused no uterine hypertrophy in ovariectomized rats compared with ovariectomized controls and compared with a doubling of uterine weight in Premarin treated ovariectomized rats. [Washburn S A et al., A conjugated equine estrogen with differential effects on uterine weight and plasma cholesterol in the rat. Am J Obstet Gynecol 1993;169:251-6]. It was determined that DHEN caused a 70% reduction in total plasma cholesterol concentrations compared with ovariectomized controls and compared with a 15% reduction of total plasma cholesterol in ovariectomized rats treated with oral Premarin.RTM..

Other Reference Publication (1):
Washburn, S.A. et al., A Conjugated Equine Estrogen With Differential Effectsd on Uterine Weight and Plasma Cholesterol In the Rat. Am J Obstet Gynecol 1993; 169:251-6.

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L7: Entry 18 of 18 File: USPT Oct 22, 1974

DOCUMENT-IDENTIFIER: US 3843662 A

**** See image for Certificate of Correction ****

TITLE: 2-HALO-5-(SUBSTITUTED PIPERIDINO SULFONYL) BENZOIC ACIDS

Brief Summary Text (3):

It has been found that those suffering from the disease exhibit elevated levels of plasma lipoprotein, of which cholesterol and triglycerides comprise major constituents. While the etiology of the disease is not yet fully understood, it is believed that .beta.-lipoproteins play an important role, and it has been recommended that dietary habits which promote low .beta.-lipoprotein plasma levels be observed. In addition, various therapeutic agents such as estrogens, thyroxine analogs and sitosterol preparations have been used to lower plasma cholesterol levels in individuals prone to the condition.

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L7: Entry 12 of 18 File: USPT Apr 23, 1996

DOCUMENT-IDENTIFIER: US 5510342 A
TITLE: Method of lowering cholesterol

Brief Summary Text (6):
Peri- and post-menopausal women are one particular group of aging persons at risk for developing coronary heart disease. Since the 1950s, it has been observed that premenopausal women are protected from coronary heart disease. These observations prompted several animal studies which demonstrated that the administration of estrogens to animals fed a high fat diet prevented dietary-induced coronary atherosclerosis. [Barrett-Connor, E., JAMA 265:(1991)]. One of the mechanisms by which estrogen is thought to be protective against atherosclerotic coronary heart disease is by lowering total plasma cholesterol (TPC) through induction of increased catabolism and excretion of low density lipoprotein (LDL) particles into bile by the liver. This increased LDL catabolism and excretion may be a result of an estrogen dependent increase in low density lipoprotein receptors in the liver, as has been demonstrated in rats given large pharmacologic doses of 17.alpha.-ethinyl estradiol. [Chao, Y-S., J. Biol. Chem. 254:11360 (1979); Kovanen, P. T., J. Biol. Chem. 254:11367 (1979); Windler E. E. T. , J. Biol. Chem., 255:10464 (1980)]. Women who receive postmenopausal estrogen replacement therapy (ERT) have been shown to benefit from a fifty to seventy percent reduction in risk from atherosclerotic related coronary heart disease. [Stampfer, M. L., N. Engl. J. Med. 313:1044 (1985)]. The mortality from CVD is 63% lower and the rate of mortality from myocardial infarction is between 2.3 and 2.7 times lower in estrogen-treated women compared with untreated climacteric women.